

mas were very small. Veit, in his text-book, had some years before noted the frequent occurrence of asthmatic attacks in young women with relatively small tumors, and indeed suggested that these attacks were of importance in the early diagnosis of myoma. Veit thought the connection between the two was through the nervous system, but all the evidence is now with Strassman and Lehmann, who refer the dyspnea to changes in the heart muscle or bloodvessels. These authors go still further and cite instances to show that the cardiac symptoms may develop before there is any suspicion of pelvic trouble. These are points of great importance, as they indicate that instead of being dependent one upon the other both the myomas and the cardiac changes must be referred to a common cause.

The presence of some intoxication to explain both immediately suggests itself, and the ovary has been selected as the possible point of its origin. The influence the ovaries exert upon metabolism is shown in the phenomena of the climacterium, in the diminished oxygen consumption after castration, which can be again increased by feeding oophorin, the increased fat deposit after removal of the ovaries, and the fact that such a serious anomaly as osteomalacia may be brought to a standstill by their removal.

These features are explained as the effects of an internal secretion, and one may assume that under abnormal conditions this altered secretion might be toxic for the heart muscle. This is, of course, all assumption and not capable of direct proof. The remarkable influence the ovaries exert over the circulatory conditions in the uterus is, of course, well known, and as myomas in their development bear such close relationship to the bloodvessels it is possible that changes in the ovaries might lead to their formation. It is of great moment then to note if the ovaries in patients with myomas are always diseased. Fleck states that gross anatomical lesions of the ovaries are always found in patients with myomas, but Kessler believes this to be an over-statement, and is convinced from his own experience and the opinions of others that in many cases the ovaries are at least macroscopically normal. He further offers as objections to the validity of the ovarian hypothesis the circumstance that enucleation of one or more myomas is seldom followed by recurrence, and that myomas may grow very rapidly and attain a large size long after the climacterium.

The clearing up of the points raised in this paper is evidently of the greatest importance in the surgical treatment of these tumors. If the ovaries are at fault the method to pursue is evident. Much valuable assistance must come from the internists who, as a rule, see these cases earlier than the surgeons, and it is particularly in regard to the early stages that statistics are needed. The character of the lesion in the heart muscle also needs to be determined. There is very little carefully collected pathological data.

The Pathogenesis of Tetany.—PINELES (*Deutsches Arch. f. klin. Med.*, 1906, LXXV, 491) in a previous communication sought to show that tetany following extirpation of the thyroid gland depends upon the destruction of the parathyroids. Clinically, no symptoms of tetany occur if these organs are left. Experimentally, animals with excised parathyroids show symptoms in every manner analogous to tetany in man. Pineles then goes very extensively into all the symp-

toms occurring in the various forms of tetany—the tetany of laborers, the tetany in acute infectious diseases, the tetany in pregnancy, the tetany in stomach disease, and the idiopathic tetany of children—analyzes them all and shows that even in minor details they are analogous to the symptoms observed in tetany strumapriya and to the symptoms of thyroidectomized animals. This remarkable similarity of all the symptoms suggests at once that a single poison is responsible for all. The tetany following the removal of the thyroid in man and animals we know is due to the destruction of the parathyroid bodies. The condition of the parathyroid in the other forms of tetany has not been definitely established.

In connection with this communication it may be well to recall the fact that McCallum in a case of fatal gastric (dilatation) tetany found the parathyroids in a condition of active hyperplasia with numerous mitotic figures, suggesting strongly the possibility that “the parathyroids had become hypertrophied to neutralize large absorption of toxin from the stomach.”—*Johns Hopkins Hospital Bulletin*, 1905, xvi, 148.

Endocarditis in Tuberculosis.—SORGO and SUEZ (*Wien. klin. Wchnschr.*, 1906, xix, 176) report at length the clinical history of a girl, aged sixteen years, who died of a slowly progressing pulmonary and laryngeal tuberculosis and amyloid disease. The condensed autopsy report reads: Chronic fibroid phthisis with cavity formation of both upper lobes; disseminated miliary and conglomerate tuberculosis in both lower and the right middle lobes; ulcerating tuberculous infiltration of the larynx and trachea; extensive tuberculous ulceration of the ascending colon; scattered tuberculous ulceration in the small intestine; microscopic tuberculous areas in the spleen, liver, and kidneys; amyloid degeneration of the spleen, liver, and kidneys; verrucose endocarditis of the mitral valve.

The endocarditis gave no symptoms during life and was not diagnosed.

Along the border of the aortic segment of the mitral valve and to a much less extent along the border of the opposite segment are numerous pale yellow, uneven, firm, warty excrescences. The largest of these measure about 2 x 4 mm. and are situated just at the free border of the valve. Their central and basal portion consists of compact bundles of spindle-shaped cells, poor in protoplasm which proceed without definite demarkation from the normal valve tissue. The periphery consists of a homogeneous eosin-staining mass indistinctly separated from the dense central portion by a more cellular layer made up of connective tissue cells rich in protoplasm and a small number of leukocytes. In the small nodules the firm connective tissue is often wanting, and they appear to be fastened directly upon the abraded epithelium of the valve. In both forms the interstices are filled with a fibrin net-work enclosing blood corpuscles, principally polymorphonuclear leukocytes, and containing numerous tubercle bacilli. None of the tissue shows any evidence of tubercle formation. Cultures from the vegetations gave a pure growth of tubercle bacilli and no other organisms were found in microscopic preparations.

Cases of true tuberculous endocarditis are rare. From a review of the literature Sorgo and Suez are willing to accept only six reported instances as genuine. The case of Braillon and Tousset is particularly